WARTHIN (A. S.)

ACCENTUATION OF THE PULMONARY SECOND SOUND AN IMPORTANT SIGN IN THE DIAGNOSIS OF PERICARDITIS.

BY

ALDRED S. WARTHIN, PH.D., M.D.,

DEMONSTRATOR OF CLINICAL MEDICINE IN THE UNIVERSITY OF MICHIGAN, ANN ARBOR.

FROM

THE MEDICAL NEWS,

April 13, 1895.

[Reprinted from THE MEDICAL NEWS, April 13, 1895.]

ACCENTUATION OF THE PULMONARY SECOND SOUND AN IMPORTANT SIGN IN THE DIAGNOSIS OF PERICARDITIS.[1]

BY ALDRED S. WARTHIN, PH.D., M.D.,
DEMONSTRATOR OF CLINICAL MEDICINE IN THE UNIVERSITY OF MICHIGAN, ANN ARBOR.

DURING the past two years the attention of Dr. Dock and myself has been frequently called to the importance of a certain physical sign in the diagnosis of pericarditis that has not received due consideration. Having met with a large number of puzzling heart-cases, in which the chief physical sign was a marked accentuation of the pulmonary second sound, either with or without the coexistence of peculiar murmurs at the base or apex of the heart, and with the entire absence of any other condition in heart or lungs to account for this accentuation, we have taken especial pains to note its occurrence and to seek some explanation. Fortunately, we were able to follow some of these cases through the development of certain phases that left no doubt as to the diagnosis; and, from the clinical experience thus gained we have been able to widen to a valuable degree our diagnostic horizon. A brief citation of a few of the cases presenting this

[1] From the Clinic of Internal Medicine, University of Michigan, Ann Arbor.

sign may serve to show its importance and to emphasize its significance in the diagnosis of pericarditis, especially in the first stages of the disease.

Case I.—Mr. A. G. W., a student, aged twenty-four years, came to the hospital on March 24, 1894, complaining of palpitation, a feeling of constriction in the chest, and shortness of breath when attempting to sing. In addition to these symptoms he had also a slight, dry cough. Three weeks before he had had slight rheumatic pains in the knees. The examination of the lungs was entirely negative. The apex-beat was of moderate strength, and was located in the fifth intercostal space, just outside the nipple-line. A faint presystolic thrill could be felt just inside of the apex-beat. The heart-dulness began at the fourth rib, extended a little to the left of the left parasternal line, and to the right as far as the median line. On auscultation a slightly rough blowing murmur, presystolic in time, was heard best just inside of the apex-beat, where the thrill was felt. The first sound at the apex was strongly accentuated, and with it there was a soft blowing murmur of slight intensity. These murmurs were constant, and had all the characteristics of valvular murmurs. The first sound at the base of the heart was very clear and strong. No murmurs could be heard at the base. The second sound was everywhere unusually strong, but in the left second interspace its character was such as to attract immediate attention. Strongly accentuated, it gave a peculiarly clangorous shock. The heart-rhythm was slightly irregular, and the radial pulse was 80, slightly irregular, small, quick, and of low tension. A diagnosis of mitral stenosis and insufficiency was made. The next day the systolic murmur could not be heard; the other signs were as before. Comment was made

at this time upon the rough character of the presystolic murmur and upon the accentuation of the pulmonary second sound, and the correctness of the diagnosis was questioned.

On March 26th the man was again examined. The physical signs were unchanged; the murmur was not altered in character by pressure with the stethoscope. On the next day the patient was again seen. Everywhere, especially at the base, there were heard two loud friction-sounds, forming with the accentuated second sound a marked canter-rhythm, one murmur occupying the systole and replacing the first sound; the second, of rougher quality, coming after the second sound. These murmurs displayed friction fremitus; were superficial; not transmitted beyond the heart-area; were increased by pressure; affected by respiration; influenced by change of position, and were not constant. The patient complained of dyspnea, dry cough, and precordial pain. At the time of examination his temperature was 102°. On March 28th he was seen in his room. There was no change, either in the symptoms or in the physical signs. On the following day he complained much of pain in the heart-region, but no change in the signs could be discovered. To relieve the pain a mustard-plaster was applied to the heart-area. The patient let this remain until a large blister had formed; but in the twenty-four hours following its application a large effusion took place, as shown by the following signs: dulness extended, in the shape of a large triangle, from the apex at the second rib in the left sternal line down to the sixth rib, two finger-breadths to the outside of the left nipple-line, and across the sternum to the fifth intercostal space a finger's breadth to the right of the sternum. The apex-beat was in the fifth intercostal space, in the nipple-line.

A small triangle of dulness, over which vocal fremitus and breath-sounds were absent, extended two finger-breadths beyond the apex-beat. The heart-sounds were distant and very faint, with the exception of the pulmonary second, which still retained its accentuated character. Friction-sounds were absent, except at the base of the heart.

From March 31st to April 7th the man's condition rapidly improved under the treatment, which consisted of the use of salicylic acid internally and of the ice-bag locally. The area of dulness decreased; friction-sounds disappeared; the heart-sounds became strong and clear. The accentuation of the second sound in the left second interspace still remained, and was the most striking sign. Repeated thorough examinations of the lungs were entirely negative. The patient remained under observation until he had made a complete recovery. The heart-dulness became normal; all murmurs and friction-sounds disappeared; the accentuation of the pulmonary sound remained for some time, but finally the sound became normal.

Case II.—Mr. A. C., a student, aged twenty-two years, was seen in November, 1893. He complained of sore-throat, pain in the joints, headache, and palpitation. The tonsils were enlarged and inflamed, showing numerous small points of white exudate. The temperature ranged from 102° to 104°. The physical examination of all the organs was entirely negative, with the exception of the heart. All of the sounds were increased, but especially was the pulmonary second sound accentuated, and strikingly ringing and metallic in quality. No murmurs or friction could be heard after careful examination. The heart-action was not greatly increased, but was irregular in rhythm. There was no enlargement of the heart-dulness. Because of the accentuation of

the pulmonary sound without other signs, special attention was paid to the heart-examination. At one visit slight friction was heard in the pulmonary area, but it disappeared, and did not return. The patient recovered without other incident; but when last seen the accentuated character of the pulmonary sound was still marked.

During the year following the man had two other attacks resembling this one, but was seen by other physicians, who treated him for "febricula." In the latter part of December, 1894, he was again taken ill with similar symptoms, and was seen on the first day by Dr. Dock, who found nothing abnormal in the heart-examination. The case then passed into other hands; and finally, because of continued fever, malaise, etc., was sent to the hospital as a suspected case of typhoid. He had also complained of pain under the sternum. When I first saw him he had no fever; the heart was slow and irregular; all of the sounds were prolonged and strongly accentuated, the pulmonary second having the same marked ringing character as when first seen a year before; it was also markedly divided, and, in addition, there were heard all over the heart, especially at the base, friction-sounds of moderate intensity. The heart-dulness was enlarged to the right, extending a finger's breadth to the right of the sternal line. The apex-beat was doubled, the second impulse being less strong than the first. The patient remained under observation for some days, during which time he had no fever; the heart-dulness became normal; the friction and the accentuation of the heart-sounds became much less marked, but were still present. Many careful examinations of the lungs were negative. As all symptoms had disappeared, the patient thought that he had entirely recovered, and so left the hos-

pital. Though advised to return for further examination, he has not reported since his discharge.

CASE III.—Mr. R. E., a student, aged twenty-eight years, came to the hospital for an examination of his heart. For a month he had been troubled with frequent attacks of palpitation and marked irregularity of the heart's action, without other symptoms. The examination of the lungs was entirely negative. The apex-beat was very strong, but in normal position. No thrill could be felt. The area of heart-dulness was not enlarged. The heart-sounds were everywhere loud and roughened, especially in the pulmonary area, where the second sound was clangorous in quality, producing a very marked shock. Here also, after slight exertion, double friction-sounds were audible, but were heard sometimes when the heart's action was quiet. The heart-rhythm was quite irregular. Several days after this he was seen again; the heart was slower and more regular; the first sound in the pulmonary area was murmurish; but the second was still strongly accentuated. There was also fine friction at the end of the systole, and sometimes in both systole and diastole. This was heard best in the third intercostal space near the left edge of the sternum. It continued when the patient suspended respiration, but sometimes disappeared altogether. The man was seen at short intervals for a month. The friction and accentuation of the pulmonary second sound entirely disappeared, but after violent exercise a soft systolic murmur was heard at the base, the heart-rhythm becoming irregular. There was no return of the symptoms, and when last seen the heart-sounds were normal.

CASE IV.—Mr. A. W., student, aged nineteen years, passed his physical examination in the gymnasium, but it was noted that his heart's action was greatly increased, and that the pulmonary sound was

"peculiar." He was told then that his heart was not normal, and that he should report once a month for examination. Becoming somewhat alarmed, he came to the hospital for an examination. He had no subjective symptoms except shortness of breath when attempting to sing. The physical examination, with the exception of that of the heart, was entirely negative. There was a strong apex-beat in the normal location, but no thrill was felt. The heart-dulness was not enlarged. Everywhere, especially at the base, there was pericardial friction. The first sound was murmerish all over, especially in the left second interspace. Here also the second sound was loudly ringing in character, causing a shock that could be felt plainly by the hand. The aortic sound was also increased, but much less intense than the pulmonary, and of entirely different quality. The next examination was made several days later. At this time there was no friction; the first sound in the pulmonary area was very murmurish, and the second still accentuated; otherwise the heart-sounds were normal. Two days afterward he was seen again. There was pericardial friction all over, the loudest and roughest sound occurring just before the strongly accentuated pulmonary sound. The first sound at the base was replaced by a soft, blowing murmur distinct from friction-sounds. For a month after this the man was examined at very short intervals. The friction-sounds varied greatly, often being entirely absent; the pulmonary accentuation and irregularity of rhythm were constant. The soft systolic murmur at the base was thought to be an anemic murmur. Nearly two months after these signs were first noted there were the physical signs of a moderate effusion. This disappeared in a short time, and since then no friction has been heard; the accentuation of the pulmonary sound is much less marked, and the

heart is more regular. The patient is still kept under observation.

Case V.—Mr. J. Q., a laborer, aged twenty-seven years, was admitted for treatment of chronic gastritis. He had no symptoms referable to heart or lungs. When examined a strongly marked accentuation of the pulmonary sound was found, with a murmurish first sound at the base. The second sound was a'so everywhere divided, the division being more marked in the pulmonary region than elsewhere. Though the aortic sound, as heard over the remainder of the heart's area, was unusually loud, it did not have the clangorous quality of the pulmonary sound. The murmurish character of the first sound disappeared when the patient assumed the erect position. No friction-sounds could be heard anywhere; the heart-dulness was not enlarged, and the rhythm was regular. From the strong accentuation of the pulmonary sound, unexplained by the existence of any other condition in heart or lungs, the suspicion of pericarditis was entertained, and the patient was advised to remain for further observation.

When examined the next day, a loud, blowing murmur was heard at the base. It resembled a valvular murmur, was transmitted upward and to the right. This was thought by one of the staff, who saw the patient for the first time, to be an aortic murmur. The second sounds were as before. Two days afterward a peculiar shuffling sound of slight intensity was heard in the pulmonary area, on pressure becoming rough and friction-like in character. For several days after this typical friction-sounds were heard at different intervals; but often they were absent, and the only sign present was the pulmonary accentuation. After being under observation for two weeks, without other signs or symp-

toms, the temperature suddenly rose to 102° or 103°, and there was marked friction all over the heart-area, with increase of heart-dulness, extending beyond the apex-beat. The pulmonary second sound retained its clangorous character. Under the salicylic-acid treatment, which we have used so largely in this condition and in pleurisy with effusion, the signs of effusion quickly disappeared; but the man still has peculiar shuffling-sounds at the base of heart, with a divided and ringing second sound in the left second interspace. For this reason he is still kept under observation.

Without going further into the detail of these cases, it will be seen that all five presented as a striking and persistent sign a very marked change in the character of the second sound as heard in the left second interspace near the sternum, a change that was not shared in by the aortic second to anything like the same degree. This change could not be explained by any condition other than the pericarditis. It began early, either with the symptoms or preceding them, and, continuing through the course of the disease, disappeared finally only after all other signs and symptoms had ceased. So far as I can remember, the sign was present in all of numerous cases of the disease that I saw in Vienna, and Dr. Dock remembers it as a prominent sign in his experience with this affection. A thorough search of the literature of pericarditis as afforded by the University library did not give much evidence as to observations made of the occurrence of this sign; and the reports of cases are almost barren so far as changes in the character of the heart-sounds are concerned. As the majority of cases reported

are those in which effusion has taken place, the chief notice taken of the heart-sounds has been in regard to their weakness, and in only few of the cases without effusion is there a mention of an accentuation of this sound. I have also been unable to find any mention of this sign in any of the special works on the heart and its diseases, and it does not find a place in the articles on pericarditis in any of the recent text-books. The French seem to have made an especial study of this disease, but many of their works in this line I have been unable to examine. So far as I have been able to find, the only notice taken of the diagnostic value of this sign is in the report of the French Congress of Internal Medicine, held in Lyons last October.

Under the head of "Les Signes de la Péricardite Aiguë," M. Josserand, of Lyons, makes this statement, which I translate entire (*La Semaine Médicale*, November 3, 1894):

In pericarditis the friction-sound is often not heard until some time after the pathologic change has begun, and Stokes asserted that it did not exist in the early stages of the disease. Nevertheless, in my opinion, there is an early sign of true value.

When, in the course of an acute articular rheumatism, one auscultates comparatively the base of the heart in the aortic and pulmonary areas, it will sometimes happen that the second sound in the latter place will be found to be much more intense, more ringing, almost clangorous, the inverse condition of that found in chronic aortitis, when the second sound is more exaggerated to the right of the sternum than to the left. Many times this same difference is appreciable to the hand, as on palpation

an exaggerated diastolic shock may be felt in the pulmonary area. The presence of this sign should lead one to search very carefully for friction-sounds, which may often be discovered when a superficial examination would have been negative; if not found, their appearance may be predicted within an interval of time of greater or less length. This accentuation of the pulmonary sound occurs early, and is very often transitory; it precedes the appearance of the friction by from one to three days. With some exceptions this accentuation disappears rather quickly, and is replaced by the friction-sound. It is a sign belonging to the initial congestive period, like the crepitant râle in pneumonia. The friction-sound, on the contrary, lasts through the period of the fever, and may be heard many weeks after the defervescence.

The pathogenesis of the accentuated sound is most probably the following: In acute pericarditis the friction-sound is most commonly heard along the left border of the sternum and in the pulmonary area. It is probable that the subjacent heart-muscle, which is near the infundibulum of the pulmonary artery, is, itself, congested and turgescent, and that this part of the myocardium, densified and covered over with a deposit of fibrin, increases the sound of the pulmonary valves, which are in close proximity. For example, there is likewise in pleurisy a slight degree of pulmonary congestion, which transmits the sounds arising in the glottis more or less modified into bronchial breathing and egophony. So there is in pericarditis a certain amount of cardiac congestion which transmits to the ear and tothe hand t he amplified sound of the pulmonary valves.

This sign has great importance with reference both to diagnosis and to treatment. In doubtful cases, when one hesitates between a friction-sound

on the one hand and an anemic or extra-cardial murmur on the other, it enables one to decide in favor of the friction. Moreover, this sign may be of interest not only in pericarditis ; endo-pericarditis is not rare. This diastolic accentuation indicates, therefore, that the heart is affected, the localization and intensity of the condition being with difficulty made precise ; and that there should be instituted early a revulsive medication, without waiting further for the later signs of murmur and friction-sounds.

While our experience would lead us to confirm M. Josserand's statements in regard to this sign, it will also permit us to add to his observations and to emphasize his conclusions in regard to its importance. It is highly probable that simple pericarditis, occurring without connection with rheumatism or other disease, is of much more frequent occurrence than is ordinarily supposed. Post-mortem statistics show this plainly. The absence of striking symptoms, the too frequently rare and hasty examinations of the heart, make it especially liable to escape recognition. As effusion may be long delayed or very slight, the presence of pericarditis may not be recognized from the slight signs and symptoms ; and the patient may often be allowed to engage in pursuits of actual harm in adding strain to a heart already working at a disadvantage. Three of our patients while in this condition were permitted to do regular gymnasium-work, although exercise produced in each very disagreeable palpitation and shortness of breath. The recognition of the disease by this sign is therefore of the greatest importance to the patient, who may be guarded from exposure and

injudicious acts, put upon suitable treatment, and carefully watched for the signs of effusion, which may come on at any time and prove to be a very serious affair. Many unexplained cases of palpitation and cardiac irregularity may possibly be made clear by recognition of this sign. Several years ago, before our attention was called to the significance of this pulmonary accentuation, we were occasionally puzzled by cases presenting these symptoms, and on physical examination exhibiting this accentuation in the pulmonary area, with peculiar and unexplained basal murmurs. It is now a question whether these were not cases of pericarditis.

This sign is certainly a very early one; but in our experience it is not transitory, being the very last thing to disappear. If the explanation of its cause as given by M. Josserand is correct, and it certainly seems adequate to me, having arrived at the same conclusion before I saw his article, it must happen that a thickening of the pericardium around the pulmonary artery or a deposit of fibrin in the same region must amplify the sounds of the pulmonary valves so long as the condition exists; and this may be present for a long time after the active disease has subsided. It is conceivable then that this accentuation must exist in all cases except the very mildest, in which the pathologic changes take place in only a very slight degree. The intensity of the accentuation of the sound will depend therefore upon the degree and character of change in the muscle and pericardium, and upon the amount and character of the exudate, whereby there is an increased conductivity of the pulmonary sound in the space

lying between the valves and the chest-wall. In adherent pericardium, and in adhesion of the pericardium to the pleura, we should expect to find a similar increase in the heart-sounds; and this was very striking in one case that I have seen.

The part of the pericardium surrounding the pulmonary artery seems to be, as M. Josserand has indicated, a favorable spot for the development of the disease; and in looking over the reports of cases one is struck with the frequency with which it is localized in this area. As to the occurrence of this sign in every case of pericarditis, it is of course evident that it will not be found in those cases in which the disease is localized in some other part of the pericardium than at the base, or in those cases in which there is but little change in the region of the pulmonary artery. But in the great majority of cases the pulmonary pericardium is affected, and consequently this sign will be found to be present. It is true that all of the heart-sounds may be increased in some of the cases, but the fact that it is the pulmonary second which is especially affected is clearly shown by the sharp localization of this accentuation in the second interspace on the left, the frequent presence of a distinct shock in this region, and the complete difference in quality between it and the aortic sound as heard elsewhere over the heart area.

The occurrence, therefore, of an accentuated second sound in the left second interspace that cannot be explained by any other condition in heart or lungs should always excite suspicion, especially if it is accompanied by murmurs resembling the so-

called anemic murmurs, but without the presence of an anemic condition to account for them. The patient should be advised accordingly, and repeated and frequent examinations of the heart should be made so long as the condition lasts. In rheumatism and all other diseases in which pericarditis may be a complication, the sign is of preëminent importance as giving early and definite warning of involvement of the heart. Moreover, by calling attention to the necessity of frequent examinations of the heart, the careful consideration of this sign may lead to the clearing up of many obscure and puzzling heart-cases. As it alone has been the constant sign in the cases of pericarditis that I have seen, the friction-sounds being often entirely absent, and many times so like valvular murmurs that it was impossible to differentiate them, I look upon it as one of the most striking and important signs of the disease, and beg leave to assert for it a prominent place in diagnosis.